5.0. POTENTIALLY ELEVATED EXPOSURES

5.1. INTRODUCTION

Certain groups of people may have higher exposures to the dioxin-like compounds than the general population. The following sections discuss higher exposures that may result from dietary habits, localized impacts, and cigarette smoking. Other population segments can be highly exposed due to occupational conditions or industrial accidents. For example, several epidemiological studies have evaluated whether elevated dioxin exposure has occurred to certain workers in the chemical industry, members of the Air Force who worked with Agent Orange, and residents of Seveso, Italy, who were exposed as a result of a pesticide plant explosion. These epidemiological studies are fully discussed in the Epidemiology Chapter of the Dioxin Health Reassessment Document (U.S. EPA, 1996) and should be consulted if further details are desired. This chapter, however, does not address occupational or accidental exposure. Instead, it focuses on elevated exposures among the general population from dietary habits such as breast feeding or high rates of fish ingestion, localized sources, or cigarette smoking.

5.2. NURSING INFANTS

Nursing infants may be exposed to dioxin-like compounds via consumption of breast milk. These compounds are deposited in the fatty tissues (i.e., adipose tissue, blood lipids, and breast milk) of the mother and may be transferred to the infant during nursing. Based on data from 1989, approximately 52 percent of U.S. mothers initiate breastfeeding with their newborn infants, and 40 percent continue breastfeeding for 3 months or longer (NAS, 1991). At 5 to 6 months of age, only about 20 percent of infants are breast-fed (NAS, 1991).

Using the estimated dioxin concentration in breast milk, the dose to the infant can be estimated as follows:

$$ADD_{infant} = \frac{C_{milk\ fat}\ f_3\ f_4\ IR_{milk}\ ED}{BW_{infant}\ AT} \qquad \text{(Eqn. 5-1)}$$

where,

ADD_{infant} = Average daily dose to the infant (pg/kg-d);
$C_{milk\ fat}$ = Concentration in milk fat (pg/g);
IR_{milk} = Ingestion rate of breast milk (kg/d);
ED = Exposure duration (yr);
BW_{infant} = Body weight of infant (kg);
AT = Averaging time (yr);
f_3 = Fraction of fat in breast milk; and
f_4 = Fraction of ingested contaminant that is absorbed.

This approach assumes that the contaminant concentration in milk represents the average over the breast feeding time period. If the dynamic models mentioned above are used, the dose can be estimated using an integration approach to account for the changes in concentration over time.

Smith (1987) reported that a study in Britain found that the breast milk ingestion rate for 7- to 8-month old infants ranged from 677 to 922 mL/d and that a study in Houston measured the mean production of lactating women to range from 723 to 751 g/d. Smith (1987) also reported that breast milk ingestion rates remain relatively constant over an infant's life, that the milk can be assumed to have a 4 percent fat content, and that 90 percent of the ingested contaminant are absorbed. The National Center for Health Statistics (1987) reported the following mean body weights for infants:

6-11 months:	9.1 kg
1 year:	11.3 kg
2 year:	13.3 kg

Using Equation 5-1 and assuming that an infant breast feeds for 1 year, has an average weight during this period of 10 kg, ingests 800 g/d breast milk, and that the dioxin concentration in milk fat is 20 ppt TEQ_{DF}-WHO_{98}, the ADD to the infant over this period (i.e., AT = 1 yr) is predicted to be about 60 pg TEQ_{DF}-WHO_{98}/kg-d [(20 pg/g x 0.04 x 0.9 x 800 g/d x 365 d) / (10 kg x 365 d) = 57.6 pg/kg-d]. This value is much higher than the estimated range for background TEQ_{DF}-WHO_{98} exposure to adults (i.e., 0.65 to 2.0 pg TEQ_{DF}-WHO_{98}/kg-d). However, if a 70 year averaging time is used, then the LADD (Lifetime

Average Daily Dose) is estimated to be 0.82 pg TEQ_{DF}-WHO_{98}/kg-d (57.6 pg/kg-d/70), which is within the range of the adult background CDD/CDF exposure range. On a mass basis, the cumulative dose to the infant under this scenario is about 210 ng (57.6 pg/kg-d x 10 kg x 365 d x ng/1,000 pg) compared to a lifetime background dose of about 1,400 to 4,100 ng (i.e., 0.65 to 2.0 pg TEQ_{DF}-WHO_{98}/kg-d x 70 kg x 365 d/yr x 69 yrs x ng/1,000 pg + 210 ng), suggesting that 5 to 16 percent of the lifetime dose may occur as a result of breast feeding. Traditionally, EPA has used the LADD as the basis for evaluating cancer risk and the ADD (i.e., the daily exposure per unit body weight occurring during an exposure event) as the more appropriate indicator of risk for noncancer endpoints. This issue is discussed further in the companion document on dioxin health effects (U.S. EPA, 1996).

The simplified procedure described above contains a number of uncertainties. A tendency toward overestimates of the dose to the infant is caused by the assumption that reductions do not occur in maternal fat levels during breast feeding. Sullivan et al. (1991) estimates that the steady-state assumption may lead to overestimates of 20 percent.

Abraham et al. (1995) studied CDD/CDF and PCB levels in the blood of a breast-fed and a formula-fed infant at 11 and 25 months of age. The body burden of dioxin-like compounds was more than an order of magnitude higher for the breast-fed infant than the formula-fed infant during both time periods (Table 5-1). A slight increase in the lipid-based CDD/CDF I-TEQ concentration in the blood of the breast-fed infant had occurred even though the child had been weaned at 10 months of age. This increase was attributed to the relative decrease in body fat mass during the period between sampling and slight increases in body burden concentrations. For the formula-fed infant, few CDD/CDFs were detected during the first sampling period, and only OCDD was detected during the second sampling period.

5.3. SPORT AND SUBSISTENCE FISHERS

The possibility of high exposure to dioxin as a result of fish consumption is most likely to occur in situations where individuals consume a large quantity of fish from one location where the dioxin level in the fish is elevated above background levels. Most people eat fish from multiple sources, and even if large quantities are consumed, they are not likely to have unusually high exposures. However, individuals who fish regularly for

purposes of basic subsistence are likely to obtain their fish from one source and have the potential for elevated exposures. Such individuals may consume large quantities of fish. U.S. EPA (1997) presents studies that indicate that Native American subsistence fishermen consume 59 g fish/day (as a mean) and 170 g fish/day (as an upper estimate). Wolfe and Walker (1987) found subsistence fish ingestion rates up to 300 g/day in a study conducted in Alaska. Assuming that subsistence fishermen consume 59 to 170 g of fresh water fish per day as their primary source of protein (i.e., no meat or eggs are consumed) adult daily intake of CDD/CDFs would be 1.3 to 3.2 pg/kg-day (Table 5-2). This estimate is based on the same CDD/CDF media concentrations exposure assumptions, and exposure algorithms as those presented in Chapter 4. The estimated values for subsistence fishermen are two to five times higher than the adult general population mean daily intake from all food sources of 0.65 pg/kg-day, as estimated in Chapter 4.

Studies are underway to evaluate whether Native Americans living on the Columbia River in Washington have high dioxin exposures as a result of fish consumption. These Tribes consume large quantities of salmon from the river. As cited in U.S. EPA (1997), a study conducted by the Columbia River Intertribal Fish Commission (1994) suggested that these individuals have an average fish consumption rate of 59 g/day and a 95th percentile rate of 170 g/day. These data were used in the estimated dietary intake calculations for subsistence fishermen, as shown above. Currently, studies are underway to measure dioxin levels in fish from this region.

Svensson et al. (1991) found elevated blood levels of CDDs and CDFs in high fish consumers living near the Baltic Sea in Sweden. Three groups were studied: nonconsumers (n=9), moderate consumers (n=9, 220 to 500 g/wk), and high consumers (n=11, 700 to 1,750 g/wk). The high consumer group was composed of fishermen or workers in the fish industry who consumed primarily salmon (30 to 90 pg I-TEQ_{DF}/g) and herring (8 to 18 pg I-TEQ_{DF}/g) from the Baltic Sea. The I-TEQ_{DF} blood level was found to average about 60 pg I-TEQ_{DF}/g lipid among the high consumers and 20 pg I-TEQ_{DF}/g lipid for the nonconsumers. This difference was particularly apparent for the PeCDFs.

Asplund et al. (1994) also found elevated plasma levels of dioxin-like PCBs in Swedish fishermen who consumed large amounts of fish. A total of 37 individuals with varying intake rates of fish from the Baltic Sea was studied. These individuals were categorized as high-fish eaters, moderate fish-eaters, and nonfish-eaters. The estimated

weekly intake of fish correlated positively with plasma PCB levels among this group (Table 5-3).

Cole et al. (1995) reported on CDD/CDFs and PCBs in 132 serum samples (pooled to 14) from Ontario Great Lakes anglers and control populations. Based on a preliminary survey, anglers from the communities of Cornwall and Mississauga, Canada, were categorized based on the numbers, species, and locations of fish caught and kept for consumption, and on data reflecting the contaminant levels for the fish in these areas. Individuals categorized as having the highest and lowest potential for having elevated body burdens of CDD/CDFs and PCBs were selected for biological sampling. Individuals who did not consume fish served as controls. Study participants were further categorized by age (i.e., <38 years, 38-50 years, and >50 years). The results, however, indicated that mean I-TEQ_{DF} levels were similar for both eaters and noneaters of Great Lakes' fish in these communities. I-TEQ_{DF}s ranged from 20.8 to 41.2 ppt for fish eaters and 24.7 to 36.8 ppt for noneaters. In general, mean I-TEQ_{DF}s increased with age (Table 5-4). PCBs 77, 126, and 169 were also evaluated in the serum samples collected from Cornwall residents. Mean TEQ_{P}-WHO_{98}s ranged from 2.6 to 17.3 ppt for fish eaters and noneaters combined. Again, significant differences between the two groups were not observed and the serum CDD/CDF and PCB levels are within the range of values observed for the general population, as presented in Chapter 4.

Health departments of five Great Lakes states: Wisconsin, Michigan, Ohio, Illinois, and Indiana, formed a consortium to study blood levels of chemical residues in fish consumers of three Great Lakes: Michigan, Huron, and Erie. Anderson et al. (1998) reported on a feasibility study to determine which compounds might be found in very frequent Great Lakes sport fish consumers. Anderson et al. (1998) selected 32 angling enthusiasts who reported eating at least one sport fish meal per week from one of three Great Lakes (i.e., 11 Lake Huron anglers, 11 Lake Erie anglers and 10 Lake Michigan anglers). The analysis included examination of serum levels of 7 CDDs, 10 CDFs, 4 coplanar PCBs (i.e., 77, 81,126, and 169), and 32 other PCB congeners. One individual was excluded from the data summary due to unusually high occupational/environmental exposures. The blood CDD/CDF/PCB levels for these anglers were compared to CDD/CDF/PCB blood levels for a comparison group (n = 70) from Jacksonville, Arkansas. Data for this Arkansas population are discussed in Chapter 4. The comparison groups

represented the general population with no known exposure to the contaminants of concern (Anderson et al., 1998). The mean CDD/CDF lipid adjusted serum concentrations for both the sport fishing populations and the comparison group used by Anderson et al. (1998) are shown in Table 5-5. The mean coplanar and other PCB lipid adjusted serum concentrations are reported in Table 5-6. The average lipid-based I-TEQ_{DFP} concentration for Great Lakes fish consumers was calculated at 56.8 ppt, with the breakdown as follows: I-TEQ_D = 27.5 ppt; I-TEQ_F = 11.9 ppt, and TEQ_P-WHO_{94} = 17.4 ppt. Anderson et al (1998) suggested that these values were higher than the background population used in the comparison.

The Anderson et al. (1998) study led to a larger study, in which the blood of 100 additional sport fishers were sampled, and a comparison population of 100 other individuals were sampled. Falk et al. (1999) reported on the results of the blood sampling from 96 (of the 100) additional sport fishers. Results for the 100 comparison population were not provided. Falk et al. (1999) presented results in terms of I-TEQ_{DF} and TEQ_P -WHO_{94} (congener-specific data were not provided), and also examined relationships between the CDD/CDF/PCB measurements in blood and factors such as: age, gender, which Great Lakes the fish came from, the type of sport fish consumed, and amount of sport fish consumed, as reported by the participants. The median lipid-based TEQ_{DFP}-WHO_{94} from the 96 participants was 21.3 ppt, with the breakdown as follows: I-TEQ_D = 9.6 ppt; I-TEQ_F = 7.4 ppt, and TEQ_P-WHO_{94} = 4.3 ppt. This finding of 21.3 ppt TEQ_{DFP}-WHO_{94} appears significantly lower than the original finding of 56.7 TEQ_{DFP}-WHO_{98}. One reason for this is that the lower finding from the 96 participants was a *median*, while the finding from the 31 individuals in the pilot study was a *mean*. It also appears that the smaller population had a few individuals with very high levels of dioxins which resulted in a higher mean concentration. Other differences that could be identified from Anderson et al. (1998) and Falk et al. (1999) include: 1) the time of sampling of the two studies; 2) the age of the participants; and 3) the sport fish consumption rates. Anderson et al. (1998) reported that sampling of the initial 31 individuals in the pilot study occurred in 1993. Although Falk et al. (1999) did not identify the date at which the followup study occurred, it appears likely to have been in 1995 or 1996. Although not expected to be a large factor explaining the differences in the populations, it is possible that average body burdens within the population decreased during this time period. The mean age of the 31 participants in the

pilot study was 52 years (range 36 to 76), while the mean age in the second population of 96 participants was 46 years (range 27-67). A clear age relationship has been demonstrated in other studies, showing that older individuals have higher body burdens of dioxins. The followup study population of 96 individuals clearly showed lower consumption of Great Lakes fish as compared to the pilot population of 31 individuals, as evidenced by questionnaire response data. The followup study population reported an average of 52 fish meals consumed per year, while the pilot group reported an average of 77 fish meals per year. Likewise, consumption of Great Lakes fish was lower for the followup group than the pilot group: 43 Great Lakes fish meals per year and 49 Great Lakes fish meals per year, respectively. Also, the followup group reported a lower number of years consuming Great Lakes sports fish (26 years) than the pilot group (33 years). In summary, while the larger population of 96 sport fishers in the full survey appeared to show a much lower body burden of dioxin-like compounds as compared to pilot population of 31 sport fishers, the differences could be explained by factors of data description (median vs. mean), year of sampling, age of participants, and exposure to dioxins in fish.

Another observation from the Anderson et al. (1998) study was that Lake Erie sport fish consumers had consistently lower CDD/CDF/PCB serum concentrations than consumers of sport fish from Lakes Michigan and Huron. Serum levels observed for the Lake Michigan and Lake Huron fish consumers were similar and higher than those observed in consumers of Lake Erie sport fish. These interlake differences parallel the pattern observed in previously reported EPA sport fish tissue monitoring data from the respective lakes (Anderson et al., 1998) and indicate that serum concentrations may also be affected by variations in fish concentrations among the lakes.

Hong et al. (1994) analyzed PCBs in human milk from Mohawk and control women to evaluate the potential effect that relatively high levels of environmental contamination may have had on the body burdens of lactating Mohawk women in New York. PCBs were found to be present in fish and wildlife in the vicinity of the Mohawk Reservation, and the Mohawk people formerly depended on local fish and wildlife for food. However, no significant differences were observed between the mean total dioxin-like PCB levels in milk from 30 Mohawk women and the 20 control women. The mean PCB concentrations for these women were 49 ppb and 55 ppb, respectively. The age of the mother, the length of the nursing period, and the number of breastfed children were found to influence PCB levels

in human milk. Older women, mothers of first born children, and smokers had higher levels of PCBs. PCB levels were also higher at the onset of lactation and in earlier samples during a breastfeeding session.

Dewailly et al. (1994) observed elevated levels of dioxin-like PCBs in the blood of fishermen on the north shore of the Gulf of the St. Lawrence River who consume large amounts of seafood. Of the 185 study samples, the 10 samples with the highest total PCB levels were analyzed for dioxin-like PCBs. Samples from Red Cross blood donors in Ontario served as controls. Dioxin-like PCB levels were 20 times higher among the 10 highly exposed fishermen than among the controls (Table 5-7). Based on these results of the 10 highest samples, Dewailly et al. (1994) estimated that for the entire fishing population studied, dioxin-like PCB levels would be eight to ten times higher than the control group. Dewailly et al. (1994) also observed elevated levels of dioxin-like PCBs in the breast milk of Inuit women of Arctic Quebec. The principal source of protein for the Inuit people is fish and sea mammal consumption. Breast milk samples were collected from 109 Inuit women within the first 3 days after delivery and analyzed for di-ortho-dioxin-like PCBs during 1989 and 1990. Subsets of 35 and 40 randomly selected samples were analyzed for mono-ortho dioxin-like and non-ortho dioxin-like PCBs, respectively. Samples from 96 Caucasian women from Quebec served as controls. The levels of non-ortho dioxin-like PCBs for Inuit women ranged from 24.7 to 220.9 ppt. These values were three to seven times higher than those observed in the control group. For mono-ortho and di-ortho dioxin-like PCBS, the levels among the Inuit women were three to ten times higher than in the control group.

5.4. LOCALIZED IMPACTS

Data have been collected that demonstrate that localized impacts may occur from emissions of dioxins from incinerators and other potential sources. "Localized impacts" are defined as measurements of CDD/CDFs in environmental (air, soil) or biotic (vegetation, animal tissue) samples near incinerators or other sources that show elevation above typical background levels for the area being studied. Therefore, "impacts," as used below, refer to elevation above background. These localized impacts may result in elevated exposure among some members of the population. Most of the data on localized impacts originate from studies conducted outside the United States, specifically from the European countries of England, Switzerland, Germany, Austria, The Netherlands, Belgium, and France. Data

collected include concentrations of dioxins in air and soil, biota including grass and cow's milk, as well as human blood and hair samples. This section reviews several of these studies, primarily discussing results in terms of TEQs from CDD/CDFs only. Following a review of the studies, the principal findings with regard to localized impacts are summarized.

Beck et al. (1990) sampled milk from a rural and an industrial area in Germany, and from dairies near a metals reclamation plant in Austria. Beck et al. (1990) observed average lipid-based concentration of 0.9 pg I-TEQ_{DF}/g in rural, background milk, 2.5 pg I-TEQ_{DF}/g in "industrial milk," and 9.6 pg I-TEQ_{DF}/g in the milk obtained from dairies near the metals reclamation plant. The dairy nearest the metals reclamation plant was located about a kilometer in the downwind direction and had the highest milk concentration (i.e., 14 pg I-TEQ_{DF}/g).

The Austrian metals reclamation plant described above has also been studied for impacts to air, soil, vegetation, and human blood by another research team (Riss et al., 1990; Riss, 1993). The plant was located in a rural Alpine river valley in Tyrol, Austria, in a mostly agriculture area. Although emissions data were unavailable, air concentrations measured near the incinerator were 1.2 to 2.3 pg I-TEQ_{DF}/m^3 (Riss, 1993). These data suggest very high emissions, because typical urban air concentrations are approximately 0.10 pg/m^3 in Europe as well as in the United States, and rural air concentrations are typically less than 0.05 pg I-TEQ_{DF}/m^3. (See Chapter 3.) Soil concentrations averaged 420 pg I-TEQ_{DF}/g at the site of the incinerator, 170 pg/g within 200 meters of plant, and 46 ppt about 2 km in the downwind direction (Riss et al., 1990). This compares with typical urban soil concentrations of approximately 10 to 20 pg I-TEQ_{DF}/g in both Europe and the United States and rural soil concentrations of less than 5 pg I-TEQ_{DF}/g. (See Chapter 3.)

A dairy farm was located between 1,400 and 2,100 meters from the same metals reclamation site in the downwind direction, and members of that farming family consumed milk from their own cows. Samples of the cows milk ranged from 20.1 to 69.5 pg I-TEQ_{DF}/g on a lipid basis. Given a general background level of milk in the low to sub ppt level on a lipid basis, it is clear that the milk showed elevated dioxin levels. (See Chapter 3.) Samples in the grass and hay from that farm were also elevated at 13 to 36 pg I-TEQ_{DF}/g dry weight. This compares to typical grass samples found in rural areas at the low to sub ppt levels (Reed et al., 1990; Kjeller et al., 1991; 1996). Blood samples from

two farmers who consumed this milk were also elevated. Their blood CDD/CDF concentrations were 152 and 946 pg I-TEQ_{DF}/g on a lipid basis. Subsequent samples from three additional family members were also slightly elevated above typical levels at 41, 66, and 77 pg TEQ_{P}-WHO_{94}/g lipid.

The Austrian samples described above were taken in the late 1980s, before emission controls and other practices (i.e, removal of some plastics) were undertaken to reduce emissions from these plants. Riss (1993) reported on reductions in both cow's milk and fodder from this nearby farm in the early 1990s and speculated that they resulted from reductions in incinerator emissions. CDD/CDF concentrations in cows' milk dropped steadily from a high in 1987/88 samplings, averaging 49 pg I-TEQ_{DF}/g fat, to an average of 5 pg I-TEQ_{DF}/g fat in the 1992/93 sampling. Grass concentrations similarly dropped from 33 pg I-TEQ_{DF}/g dry weight to 4 pg I-TEQ_{DF}/g dry weight between the two sample dates. This trend demonstrates an important expectation with regard to environmental responses to reductions in emissions from tall industrial stacks. Specifically, vegetation appears to respond immediately to reduced air concentrations, and if dairy cows are being fed with vegetation that has reduced concentrations, cow's milk should similarly respond in a rapid manner. Fries and Paustenbach (1990) stated that a steady state is reached in cow's milk with a constant dietary input of dioxins after about 30 to 60 days. Therefore, reductions in emissions will result in both a reduction in vegetation and cow's milk concentrations almost simultaneously.

Another study was conducted in Austria by Moche and Thanner (1997). The study evaluated ambient air patterns and CDD/CDF concentrations in a vicinity of steel production plants in Leoben/Donawitz. Samples were collected from sites in the immediate vicinity of the production plants, in an area that was expected to be impacted by the production plants, and in an area that was shielded by mountains in the northwest. Sampling occurred over four periods to address the potential influence of the summer and winter fluctuations in CDD/CDF concentration. The CDD/CDF concentrations in these samples were compared to previous data collected in the three Austrian conurbations Graz, Linz, and Wien. The previous data suggested average summer levels of CDD/CDFs in the range of 20 to 40 fg I-TEQ_{DF}/Nm^3 and winter levels in the range of 50 to 220 fg I-TEQ_{DF}/Nm^3. The data collected at Leoben/Donawitz indicated higher ambient air levels of CDD/CDF concentrations. Only the levels in the area shielded by mountains fall within the levels of the previously reported

data. In addition, the CDD/CDF profiles of the Leoben/Donawitz sites indicated a high contribution of the lower chlorinated CDFs (tetra- through hexachlorinated CDFs as the most abundant). The patterns were in good agreement with emission profiles of metallurgical processes reported by Hagenmaier et al. (1994) (Moche and Thanner, 1997).

Liem et al. (1991) reported on the analysis of over 200 samples of cow's milk that were taken in various regions in The Netherlands, including some that were near municipal solid waste incinerators and metals reclamations plants, and some identified as background sites. Background levels ranged from 0.7 to 2.5 pg I-TEQ_{DF}/g lipid. The highest levels were found approximately 2 km from the largest municipal solid waste incinerator identified at the time, with concentrations ranging from 2.8 to 12.6 pg I-TEQ_{DF}/g lipid. Higher than background levels were also found in samplings near other incinerators. The researchers did a principal component analysis on congener profiles in the milk samples to determine if there were any discernable differences among groupings of samples. Liem et al. (1991) observed a distinct pattern for samples around the metals reclamation plant compared to samples around municipal solid waste facilities. A higher CDF/CDD ratio was found around the metals reclamation plant (i.e., higher furan concentrations were in the milk near the metals reclamation plant than near the municipal solid waste incinerator). Liem et al. (1991) speculated that metals reclamation plants process cables that contain PVC, and according to Christmann et al. (1989), furans are predominantly formed in the combustion of PVC. Subsequently, the higher levels of furans would be taken up into vegetation and then into cow's milk. Liem et al. (1991) also found distinct patterns in samples associated with other facilities, as characterized by the relative amounts of lower and higher chlorinated congeners. Two of the incinerators were closed in April of 1990, and a marked decrease in sample concentrations associated with these two incinerators was noted between the February and August 1990 sampling. This supports the expectation described above regarding the response of vegetation and milk to changes in nearby source emissions.

A limited sample from six cows in Switzerland showed similarly elevated CDD/CDFs in association with incinerators or manufacturing sites. Higher CDD/CDF concentrations were observed in milk samples that were within 1,000 meters of an incinerator (two samples) and those that were within 1,000 meters of a production site for various chlorinated samples (one sample) than samples from a background farm (one sample) and

from local dairies that pooled milk from several farms (two samples) (Rappe et al., 1987). Insufficient information was available in this report to calculate I-TEQ_{DF} concentrations.

De Fre and Wevers (1998) evaluated paired CDD/CDF deposition and cow's milk data from several locations in Belgium to evaluate the relationship between deposition rates and milk levels, and the potential impact that elevated deposition rates may have on local milk supplies. CDD/CDF deposition ranged from approximately 2 ng I-TEQ_{DF}/m^2/year to 45 ng I-TEQ_{DF}/m^2/year, and CDD/CDF concentrations in milk fat ranged from approximately 1 pg I-TEQ_{DF}/g to 19 pg I-TEQ_{DF}/g. The correlation coefficient (R) for CDD/CDF deposition rates and milk fat concentrations was 0.69. The results of a regression analysis using these data indicated that milk fat concentrations of I-TEQ_{DF}s could be predicted from deposition rates using the equation $y = 0.3332x$, where y is the milk fat concentration of CDD/CDFs in units of pg TEQ_{DF}/g and x is the CDD/CDF deposition rate in units of ng TEQ_{DF}/m^2/y.

In France, the Ministry of Agriculture and Fisheries investigated CDD/CDF concentrations in cow's milk sampled from farms in a downwind direction within 11 km, but mostly within 5 km, of 26 industrial facilities (Defour et al., 1998). These industries included: steel manufacturing, secondary lead and aluminum smelting, copper refining, chemical and oil refining industries, electricity production, and municipal waste incinerators. Of the 49 milk samples analyzed, 46 samples had CDD/CDF concentrations that were less than 3 pg I-TEQ_{DF}/g fat with an average of 1.53 pg I-TEQ_{DF}/g on milk fat basis. One milk sample collected from a site near a chemistry industry was found to contain 3 to 5 pg I-TEQ_{DF}/g fat, and two milk samples collected 250 m and 1 km downwind of incinerators had concentrations higher than 5 pg I-TEQ_{DF}/g fat. The average concentration in milk and diary products in France assessed through a 1996 survey conducted by the Ministry of Agriculture and Fisheries was 1.33 pg I-TEQ_{DF}/g fat (Defour et al., 1998).

Abraham et al. (1998) reported on the levels of CDD/CDFs in the human milk of 10 mothers who lived within a radius of 8 km of Ilsenburg, Germany. The town was identified as an area highly contaminated with CDD/CDFs reportedly resulting from emissions from a copper plant. At the time of sample collection (i.e., 1997) the plant had been closed for approximately 6 years, Abraham et al. (1998) compared the findings to the results of a previous study of human milk levels conducted in 1990/1991 when the plant was still in operation. The 1990/1991 human milk samples contained a mean I-TEQ_{DF} of 59 ppt, lipid

based (n = 9). The 1997 human milk samples contained a mean I-TEQ_{DF} of 41 ppt, lipid based (n = 10). Abraham et al. (1998) documents that this decrease in CDD/CDFs is lower than the decline reported in general background concentrations in human milk from Western Germany in recent years. These values are somewhat higher than the values reported in Chapter 4 for the general population of the United States.

An extensive study was undertaken in the Pontypool environment of South Wales (Ball et al., 1993; Ball et al., 1994a; Ball et al., 1994b; Ball et al., 1995). Evidence of the impact of emissions from waste incineration at Rechem International Ltd. (a chemical company) prompted extensive investigations into impacts from emissions of PCBs and CDD/CDFs to nearby and regional media including soil, grass, water, air, fruit/vegetables, cow's milk, duck meat, and eggs from chicken and ducks. The region has a combination of residential and industrial uses, with very little agricultural uses. The greatest impact was found at a residence adjacent to the site, located only about 100 meters away. The soil at Rechem International averaged 810 pg I-TEQ_{DF}/g (n = 4), while at this nearby residence, the concentration averaged 66 pg I-TEQ_{DF}/g (n = 8). Other areas evaluated ranged from 4 to 24 pg I-TEQ_{DF}/g. Data were not available on CDD/CDF emissions, but air measurements at this residence suggested high emission rates. For five air samples taken at the residence, air concentrations ranged from 1.6 to 14.8 pg I-TEQ_{DF}/m^3. This compares to air concentrations ranging from 0.02 to 0.68 pg I-TEQ_{DF}/m^3 taken from a site about 2,500 meters away in the same direction from the Rechem site. The researchers also compared these air concentrations to average air concentrations ranging from 0.21 to 0.67 pg I-TEQ_{DF}/m^3 in four other UK urban areas. Concentrations of CDDs/CDFs in grass were found to be elevated at the same residence, but described as more typical for other grass sampling sites. Perhaps most importantly, samples of duck and bantam eggs from this residence showed concentrations that exceeded other duck and bantam egg samples in the area by a factor of 10. Duck and bantam egg concentrations in the area, but not at this residence, were described as typical of background. Duck meat at the impacted residence was not described as elevated compared to duck meat from nearby settings. Sampling of sediments in a nearby reservoir did not indicate elevated concentrations of dioxins or PCBs. There was no sampling of human blood or tissue. However, a simple exposure exercise showed that consumption of duck eggs, duck meat, apples, inhalation, and incidental soil ingestion at this impacted residence would result a daily intake of 165 pg I-TEQ_{DF}/day,

compared to a background intake from these pathways of 43.2 pg I-TEQ_{DF}/day (consumption rates described as typical derived from consumption data from the Ministry of Food and Fisheries in the UK).

Foxall et al. (1997) also reported geographical variations in environmental levels and human exposure to CDD/CDFs and PCBs of the above study. The data indicated a particular impact in a 200-meter wide strip of land around the boundary of the incineration plant owned by Rechem International Ltd. This location is predominantly downwind from the incinerator and there had been evidence suggesting that fugitive emissions from the plant contributed to the environmental impacts. Marked differences were noted between the CDD/CDF and PCB content of samples (i.e., air, soil, and foods) collected at the impacted site and those collected at rural background locations. Intakes of CDD/CDFs (pg I-TEQ_{DF}/day) and PCBs (μg/day) were estimated using mean daily food consumption rates, inhalation and soil ingestion rates of 20 m^3/day and 100 mg/day, respectively, and the median concentrations of CDD/CDFs and PCBs found in the samples. These estimates indicated that exposure to CDD/CDFs and PCBs at the impacted site was much higher than for background levels and the main contributors to these higher levels were residues in bantam and duck eggs. The estimated intake of CDD/CDFs from ingestion of bantam or duck eggs at the impacted site were 204 pg I-TEQ_{DF}/day and 103 pg I-TEQ_{DF}/day, respectively; levels that are substantially higher than the average UK dietary intake of 88 pg I-TEQ_{DF}/day from all food sources. Based on a body mass of 60 kg, these egg intakes (i.e., 3.4 and 1.7 pg I-TEQ_{DF}/kg body mass/day) would represent 34 and 17 percent of the WHO (World Health Organization) TDI (Total Dietary Intake) value of 10 pg I-TEQ_{DF}/kg body mass. Similarly, the corresponding PCB intake of 7.3 and 6.3 μg/day would represent 73 and 63 percent, respectively, of an average dietary intake (10 μg/day) of PCBs.

Lovett et al. (1998) performed additional analysis of chicken, bantam, and duck eggs; and also duck meat collected from the vicinity of the Rechem incinerator and compared the results to PCB and CDD/F levels of comparable foodstuffs collected from rural areas in the same Welsh district. Poultry produced at the impacted residence displayed a congener profile with noticeable variations compared to those collected from nearby rural sites. A prominence of higher chlorinated congeners in the egg and duck meat samples for the residence located near the incinerator was observed. Analysis of 46 PCB congeners resulted in a median fresh mass total PCB concentration in duck eggs of 191

μg/kg (n = 2), 341 μg/kg in bantam eggs (n = 2), and 43 μg/kg in duck meat (n = 2) from samples collected in the impacted area. Observations from rural areas showed fresh mass based total PCB concentrations of 14 μg/kg for duck eggs (n = 6), 22 μg/kg for bantam eggs(n = 4), and 25 μg/kg for duck meat (n = 6).

A second location in the United Kingdom, the Derbyshire area in central England, has shown elevations in cow's milk and other animal tissues. Initially, samples of cow's milk were taken by the Ministry of Agriculture, Fisheries, and Food (MAFF) from individual farm tanks on 11 farms in 1990. When 2 of the samples showed high concentrations of 40 and 42 ng I-TEQ_{DF}/kg fat (the other 9 showed more typical concentrations in the 1.1 to 7.1 ng I-TEQ_{DF}/kg fat), the sampling was expanded to 30 farms. These original two farms, plus an additional farm, continued to show high concentrations in the milk. Testing continued in milk through 1994. Milk concentrations dropped at one farm, but overall concentrations appeared to remain high (i.e., 29 ng I-TEQ_{DF}/kg) for the most recently reported sampling in July of 1994 (Harrison et al., 1996). As a result of these findings, MAFF tested animal tissue from the three farms. Calves from one of the three farms had extremely elevated levels of dioxins and furans, with concentrations ranging from 2.5 to 6.9 ng I-TEQ_{DF}/kg whole weight (i.e., not lipid basis) in muscle tissue (MAFF, 1992a). This compares, for example, with I-TEQ_{DF} concentrations approximately 0.20 ng I-TEQ_{DF}/kg in the United States beef supply (from the national study on beef back fat, assuming 19 percent fat in whole beef (Winters et al., 1996). Egg samples were taken from one of the three farms and a second "free range" supplier. The concentrations found were reported as 2.2 and 2.1 ng I-TEQ_{DF}/kg in whole eggs. A second sample from one of the farms taken a year later showed a lower concentration of 0.8 ng I-TEQ_{DF}/kg whole weight. These would appear to be elevated, considering that eggs found in a background setting in Mississippi (Cooper et al., 1995), had concentrations less than 0.10 ng I-TEQ_{DF}/kg whole weight.

MAFF (1992b) also sampled leafy herbage (grass, hay, etc.) from the three farms described above. Concentrations ranged from about 2 to 14 ng I-TEQ_{DF}/kg dry weight. This appears elevated, considering that samplings of background grass in England showed 0.89 ng I-TEQ_{DF}/kg dry weight, as reported in 1991 (Kjeller et al., 1991) and 0.57 ng I-TEQ/kg dry weight in 1996 (Kjeller et al., 1996). The evidence suggests that the impacts were due to nearby industrial emissions from a fuel plant and chemical waste incineration. Her Majesty's Inspectorate of Pollution (HMIP) conducted additional studies to evaluate this

possibility. These included stack testing of the Coalite Fuels, Ltd. and the Coalite Chemicals, Ltd. (which were adjacent to one of the farms and near the other two), air dispersion modeling, and soil monitoring. No results were available to evaluate the stack testing, but the air dispersion modeling predicted that the three impacted farms would be in the sectors having the highest air concentrations. The soil sampling on the three farms showed concentrations ranging from 10 to 90 ppt. This can be considered elevated above typical rural background and in the range or even higher than typical urban concentrations. Specifically, this compares with typical urban soil concentrations of 10 to 20 pg I-TEQ_{DF}/g in both Europe and the United States, and rural soil concentrations typically less than 5 pg I-TEQ_{DF}/g. (See Chapter 3.) Also, these concentrations are similar to concentrations found near incinerators emitting very high concentrations of dioxins in Tyrol, Austria, as described above, and in Columbus, Ohio, as described below. The National Rivers Authority sampled sediment upstream and downstream of the effluent discharge pipe of the Coalite Chemicals, Ltd site. Samples collected about 1.5 km downstream of the discharge site had CDD/CDF levels that were 1,000 times greater than background samples collected 1.5 km upstream of the discharge point (these studies reported in MAFF, 1992a).

Another interesting finding associated with the samplings of foods and environmental media in the Derbyshire area were the congener profiles. Compared to background milk samples, the samples from the three impacted farms had proportionally higher concentrations of lower chlorinated dioxins, particularly 2,3,7,8-TCDD and 1,2,3,6,7,8-HxCDD. The background samples of milk tended to be dominated by OCDD (MAFF, 1992b). Blood samples were also collected from residents of these three impacted farms and analyzed for CDD/CDFs (Startin et al., 1994). The I-TEQ_{DF} concentrations for the 10 individual blood samples ranged from 49 pg/g to 291 pg/g on a lipid basis. In contrast, the two control samples had I-TEQ_{DF} concentrations of 16 pg/g and 26 pg/g on a lipid basis. The Derbyshire samples were dominated by OCDD, followed by 1,2,3,4,6,7,8-HpCDD, 2,3,7,8-TCDD, and 1,2,3,6,7,8-HxCDD.

Sandalls et al. (1998) analyzed soil concentrations around the site of a chemical waste incinerator near Bolsover, Derbyshire, United Kingdom. At each of 46 sites, five surface soil samples were collected, at varying depths up to a depth of 5 centimeters, every 1 square meter. All 46 sample sites had total TCDD concentrations exceeding background concentrations. Higher concentrations of TCDD were observed at locations

closer to the incinerator and there was a strong correlation between the TCDD concentration in a given quadrant and the amount of time that the wind was blowing in that direction. At four quadrants around the site, TCDD soil concentrations were reported as 603 ppt for the northeast (approximately 42 percent of the total deposition); 315 ppt for the southeast (22 percent of the total); 269 ppt for the southwest (19 percent of the total); and 244 ppt (17 percent of the total) for the northwest. The results of the spatial distribution of CDD/CDFs implicated the incinerator as the likely source, and the correlation between deposition and wind direction suggested that these compounds reached the ground via the atmosphere. Also, 42 of the 46 sample sites showed similar CDD/CDF congener ratios to the flue gas of the waste incinerator. Soil concentrations were well in excess of background concentrations, up to 5 kilometers around the site.

Ohta et al. (1997) studied levels of CDD/CDF and non-ortho coplanar PCBs in soil at a high cancer-rate area close to a batch-type municipal solid waste (MSW) incinerator in Japan. Sixty-one soil samples were collected around the MSW incinerator. Among them, 52 samples were radially collected within 2 km from the center of the MSW incinerator, and 9 samples were collected across the high cancer-rate area. High concentrations of CDD/CDFs and coplanar PCBs were observed in all the soil samples from the leeward side of the MSW incinerator. Total concentrations ranged from 5,303 to 32,167 pg/g; mean = 13,934 pg/g. On the other hand, all but one sample on the windward site showed high contamination. Among the 61 samples analyzed, the total concentration was greater than 2,000 pg/g in 45 of the 61 samples and the TEQ_{DFP}-WHO_{94} concentration was over 10 pg/g in 39 of the 61 samples. In addition, the levels of CDD/CDFs and coplanar PCBs at a distance of 0 to 1.1 km from the MSW incinerator was compared with that of the area 1.1 to 2.0 km from the MSW incinerator. The area closer to the incinerator contained a higher ratio of samples with contamination over 5,000 pg/g (63.2 percent) than in the area further away from the MSW incinerator (38.5 percent). Similarly, the area closer to the incinerator had a higher percentage of samples with CDD/CDF/PCB levels over 30 pg TEQ_{DFP}-WHO_{94}/g (26.3 percent) than the area further away from the incinerator (15.3 percent).

Miyata et al. (1998) collected blood samples from residents living within 2 km from a batch-type municipal solid waste incinerator in Japan where soil concentrations of CDD/CDFs and PCBs were shown to be elevated. Eighteen blood samples were collected from 13 men, aged 23 to 63 years old (average age = 45 years), and 5 women, aged 30

to 72 years old (average age = 46 years) in March 1996. The results indicated that the average lipid-based TEQ_{DFP}-WHO_{94} concentrations found in blood samples ranged from 34 pg/g to 200 pg/g with a mean of 81 pg/g for men, and from 22 pg/g to 463 pg/g with a mean of 149 pg/g for women. These mean TEQ_{DFP}-WHO_{94} values are higher than those reported for the general population of various countries (the mean value estimated in Chapter 4 of this document is 55 pg/g).

Local impacts around a waste-to-energy municipal solid waste incinerator in Columbus, Ohio, were undertaken by the Ohio Environmental Protection Agency, and the U.S. Environmental Protection Agency (Lorber et al., 1998). This incinerator operated between 1983 and 1994. A stack test was taken in 1992, and when the results were extrapolated to typical operation of the incinerator, annual emissions were calculated at 985 g I-TEQ/yr. This is a very high emission rate, and compares to total emissions from several European countries. It is about one-tenth of the national emissions estimated for all United States sources. (See Volume II.) Process modifications were undertaken in the winter of 1993/94. A stack test was conducted which indicated that annual emissions were reduced to 267 g $I\text{-}TEQ_{DF}$/yr. The U.S. EPA undertook a soil testing program in December of 1995. Results showed a definite impact to soils at the site of the incinerator, with an average concentration of 356 pg $I\text{-}TEQ_{DF}$/g (n = 4). Of the four samples collected, three of the samples averaged 458 pg $I\text{-}TEQ_{DF}$/g and the fourth was much lower at 50 g $I\text{-}TEQ_{DF}$/g. Just offsite in the downwind direction, a cluster of four samples within 1,000 meters also showed some elevation of CDD/CDFs with an average concentration of 49 pg $I\text{-}TEQ_{DF}$/g. Fourteen additional samples, generally within 2 miles of the site, averaged 10 pg $I\text{-}TEQ_{DF}$/g. Three soil samples at a background site 28 miles away in the upwind direction averaged 1 pg $I\text{-}TEQ_{DF}$/g. These latter two clusters of urban and background samples have concentrations that are typical of urban and background situations. The urban results suggests that, despite large emissions from this source, soil impacts above typical levels appeared to be restricted to within 1,000 meters of the incinerator.

The Ohio EPA conducted air monitoring in 1994 and 1995 (OEPA, 1994; Lorber et al., 1998). Monitoring in 1994 occurred after process modifications were undertaken to reduce dioxin emissions. A stack test conducted just prior to the air sampling showed reductions of 75 percent from the levels measured in 1992. Wind rose data were taken on an hourly basis during the 1994 sampling. This showed that two samples from a sampler

located about 2 miles away were in the downwind direction during the 48-hour sampling period. Eight other samples from four samplers (which were between 1 and 2 miles from the incinerator) were clearly not in the downwind direction. The two downwind samples averaged 0.26 pg I-TEQ/m^3 CDD/CDFs, while the eight upwind samples averaged 0.05 pg I-TEQ_{DF}/m^3. The incinerator shut down in December 1994. Five samples taken in 1995 showed an average of 0.05 pg I-TEQ_{DF}/m^3 CDD/CDFs. This air sampling suggests the following: (1) the typical background urban air CDD/CDF concentration in Columbus is probably around 0.05 pg I-TEQ_{DF}/m^3; and (2) when the incinerator is emitting dioxins typical of the rate measured in the 1994 stack test (not the 1992 stack test), air concentrations about 2 miles away are higher, perhaps by a factor of 5. Other media, including vegetation, agricultural products, or human blood were not sampled.

Schecter and Papke (1998) examined CDD/CDFs and PCBs 77, 126, and 169 in blood sampled from 10 residents living near a PCB manufacturing facility in Alabama in 1997, and compared these concentrations to a pooled sample from a control group representing 100 adults. The results showed that, for the 10 residents, total lipid-based CDD/CDF concentrations ranged from 825 ppt to 6,422 ppt. The corresponding total CDD/CDF concentrations from the pooled control sample was 1,112 ppt. Total PCB concentrations ranged from 240 ppt to 5,216 ppt for the 10 nearby residents, compared to 1,112 ppt for the pooled control sample. PCB-77 was only detected in the blood of 5 of the 10 residents, ranging from 41 ppt to 713 ppt. PCB-126 concentrations ranged from 104 ppt to 4,050 ppt in residents, compared to 48 ppt found in the control sample. PCB-169 concentrations ranged from 136 ppt to 2,807 ppt for the 10 residents, compared to 35 ppt for the control sample. In terms of TEQ, the total I-TEQ_D and I-TEQ_F concentrations ranged from 16.3 ppt to 38.9 ppt, and from 6.7 ppt to 131 ppt, respectively, compared with 18.5 ppt and 8.3 ppt from the control blood. TEQ_P-WHO_{94}s ranged from 34 ppt to 360 ppt compared to 32 ppt for the control blood.

There were also studies conducted in Asia to address localized impacts. Luksemburg et al. (1997) reported, in a preliminary study, that high levels of CDD/CDFs were observed in soil and sediment samples collected inside and outside a sodium pentachlorophenate plant in Tianjin, China. The plant is situated in a wetland with rivers emptying into the nearby Pacific Ocean and close to several large housing developments. Human hair samples collected from barber shops in the housing developments near the

plant were also collected and CDD/CDFs were detected in these samples. The I-TEQ_{DF} concentrations in soil ranged from 15 ppt at a site upstream of the plant to 740,000 ppt within the plant, and was 1,800 to 2,200 ppt at sites outside the plant. The I-TEQ_{DF} concentrations in sediment ranged from 150 ppt at a site 50 km away from the site to 110,000 ppt in a drainage canal located just southwest of the plant. Hair samples contained I-TEQ_{DF} concentrations ranging from 12 to 120 ppt. According to Luksemburg et al. (1997), "the isomer profiles of all the samples were consistent with the pentachlorophenol sources." However, it should be noted that no background information on the test subjects (time of residence, health records, etc.) were collected in this study.

The major findings and conclusions based on this review of localized sources include:

- Localized impacts, meaning elevated concentrations of CDD/CDFs above background, have been found in the vicinity of some CDD/CDF sources.

- Localized impacts appear to be limited to an area within 5 km of an incinerator source, perhaps only within 2 to 3 km of the source, and in some cases, only within a few hundred meters of the source. One study noted elevations in grass, cow's milk, and human blood on a farm located 2 km from an incinerator presumed to be emitting high amounts of CDD/CDFs. Not all of the studies described in the literature discussed distance from the source, as the surveyed areas were simply identified as "industrial."

- Several studies continued environmental samplings after efforts were made to reduce emissions or after the sources were shut down. In these cases, reductions in CDD/CDF concentrations were noted for various media including, cow's milk, vegetation, and air. As discussed below, vegetation has been found to respond rapidly to reductions in air concentrations, and the time to reach steady state in cow's milk given a steady input of CDD/CDFs is also relatively short. In other cases, such as in the accumulation of CDD/CDFs in soils or in body fat, the benefits may not be as immediate. Soil

and body fat are reservoirs in which the residence time of these compounds are measured on the order of years.

The available data reviewed above suggest that measurable impacts near incinerators only occur if the incinerator emits very high amounts of dioxins, in contrast to emissions that are known to be within regulatory limits. However, two key descriptors here, including "measurable impacts" and "very high amounts of emissions" cannot be rigorously defined. The data suggest that "measurable" impacts can be defined as elevations in dioxin concentrations in environmental or biotic media on the order of 5-10 times higher than typical background. "Very high amounts" of releases is less well defined. The Columbus incinerator is the only incinerator reviewed above for which emission data were available, and the stack test of 1992 showed emissions that were about 200 times higher than the 1995 proposed regulatory limit for solid waste incinerators of 30 ng of total dioxins per m^3. By comparison of environmental media sampling, one could surmise that the metals reclamation plant in Tyrol, Austria, and the incinerators in the Derbyshire area of Central England, if not others noted above, were also emitting unusually high amounts of dioxins.

Also, it is important to understand that elevations in air, soil, vegetation, and animal products do not automatically translate to higher exposure levels. This document (and several other efforts worldwide) have concluded that the bulk of exposure to dioxins occurs via the diet, and specifically animal fats. Higher exposure to an individual would only result if an individual subsisted on animal food products from animals raised near incinerators (meaning also that the animal's diet was comprised of vegetation grown where the animal is raised), and perhaps only incinerators emitting high amounts of dioxins. As described above, there are limited human tissue data for individuals where localized environmental contamination has been demonstrated. In one study in Austria of a farming family consuming home-grown milk near the metals reclamation plant, both the milk and the blood of the family were shown to have elevated levels of dioxins. In the vicinity of a municipal solid waste incinerator in Japan where CDD/CDF and PCB concentrations in soil were elevated, nearby residents also had elevated blood levels of CDD/CDF/PCBs. In the U.S., there have been no studies to evaluate the prevalence of subsistence behavior near sources, in general, and near high emitting incinerators, in particular. Even if there is a

sparsity of subsistence behaviors near sources in the U.S., it is reasonable to assume that animal fats produced near high emitting incinerators would likely have elevated CDD/CDF levels and be consumed. If incinerators are meeting regulation limits, they would not be high emitters, and the likelihood of localized impacts would be small.

5.5. CIGARETTE SMOKERS

As discussed in Volume II, cigarette smoking has been found to be a source of CDD/CDFs. As a result, individuals who smoke cigarettes, and nonsmokers who are exposed to second-hand smoke, may experience higher levels of exposure to dioxin-like compounds than the general population. Matsueda et al. (1994) reported that the mean I-TEQ_{DF} content of a pack of U.S. cigarettes was 8.6 pg. This estimate is based on analytical data from seven brands of U.S. cigarettes. Assuming that a pack of cigarettes contains 20 cigarettes, the I-TEQ_{DF} content of a single cigarette would be 0.43 pg. This value represents about half of the I-TEQ_{DF} value reported for a mainstream cigarette smoke from a Swedish brand of cigarettes (Löfroth and Zebühr, 1992) and is about five times higher than the I-TEQ_{DF} level in mainstream smoke from German cigarettes (Ball et al., 1990). The daily intake of CDD/CDFs by smokers can be estimated by multiplying the CDD/CDF content of a single cigarette by the mean number of cigarettes smoked per day by current smokers. According to U.S. EPA (1992), 25.5 percent of the adult U.S. population were smokers in 1990. The average daily number of cigarettes smoked by this population was 19.1. Thus, mean CDD/CDF exposures via cigarette smoking are estimated to be 8.2 pg I-TEQ_{DF}/day for smokers. This level of exposure represents over 10 percent of the average daily background dose of CDD/CDFs from soil, air, water, and foods, as described in Chapter 4. The use of data on the total I-TEQ_{DF} content of a cigarette from Matsueda et al. (1994) results in uncertainties as to the estimate of exposure to smokers because the approach assumes that all of the dioxin in the unburned cigarette is inhaled. It is likely that some of the dioxins are released with the sidestream smoke rather than being inhaled. It is also possible that dioxins are destroyed and/or formed during the combustion process. Thus, it is unclear if these factors would lead to a net increase or decrease in the amount of dioxins inhaled. However, as described above, the I-TEQ_{DF} value reported by Matsueda et al. (1994) is less than that in mainstream (i.e., inhaled) smoke reported by

Löfroth and Zebühr (1992) and greater than that of Ball et al. (1990) and provides the best estimate of CDD/CDFs in cigarettes to which smokers may be exposed.

Nonsmokers may also be exposed to CDD/CDFs from environmental tobacco smoke. Although the data on the frequency, magnitude, and duration of exposure to environmental tobacco smoke are limited, an idea of the magnitude of exposure to CDD/CDFs can be gained by assuming that nonsmokers receive a fraction of the CDD/CDF TEQ received by smokers. Based on data for nicotine, the dose to nonsmokers exposed to environmental tobacco smoke is estimated to be 0.1 to 0.7 percent that of smokers (U.S. EPA, 1992). For 4-aminobiphenyl, nonsmokers exposed to environmental tobacco smoke were estimated to receive a dose that was 10 to 20 percent that of smokers (U.S. EPA, 1992). Assuming that nonsmokers receive 0.1 to 20 percent of the dose of CDD/CDFs from second-hand smoke that smokers receive, the estimated daily dose of CDD/CDFs for nonsmokers would range from 0.008 pg I-TEQ_{DF}/day to 1.6 pg I-TEQ_{DF}/day. It should be noted, however, that individual exposure to sidestream smoke is highly variable, depending on a person's proximity to smokers, how often they are near smokers, and the ventilation rate in these areas.

5.6. SUMMARY OF POTENTIALLY HIGH EXPOSURES THAN BACKGROUND LEVELS

As discussed in Chapter 4, background exposures to dioxin-like compounds are likely to extend to levels up to three times the average. This upper range is assumed to result from the normal variability of diet and human behaviors. Exposures from local elevated sources or exposures resulting from unique diets may be in addition to this background variability. Such elevated exposures may occur in small segments of the population such as individuals living near discrete local sources, or subsistence or recreational fishers. Nursing infants represent a special case where, for a limited portion of their lives, these individuals may have elevated exposures. Cigarette smokers may also experience elevated exposure levels that are elevated above the background levels estimated in Chapter 4.

CDD/CDF contamination incidents involving the commercial food supply have occurred in the U.S. and other countries. For example, in the U.S., contaminated ball clay was used as an anticaking agent in soybean meal and resulted in elevated dioxin levels in some poultry and catfish. This incident involved less than 5% of the national poultry production and has since been eliminated. Elevated dioxin levels have also been observed in

a few beef and dairy animals where the contamination was associated with contact with pentachlorophenol treated wood. Evidence of this kind of elevated exposure was not detected in the national beef survey. Consequently its occurrence is likely to be low, but it has not been determined. These incidents may have led to small increases in dioxin exposure to the general population. However, it is unlikely that such incidents have led to disproportionate exposures to populations living near where these incidents have occurred, since, in the U.S., meat and dairy products are highly distributed on a national scale. If contamination events were to occur in foods that are predominantly distributed on a local or regional scale, then such events could lead to highly exposed local populations.

Elevated exposures associated with the workplace or industrial accidents have also been documented. U.S. workers in certain segments of the chemical industry had elevated levels of TCDD exposure, with some measurements in the hundreds of ppt. There is no clear evidence that elevated exposures are currently occurring among U.S. workers. Documented examples of past exposures for other groups include certain Air Force personnel troops exposed to Agent Orange during the Vietnam War and people exposed as a result of industrial accidents in Europe and Asia. However, studies involving these populations have not been presented in this chapter.

Consumption of breast milk by nursing infants may lead to higher levels of exposure as compared to non-nursing infants and intake in the diet later in life. As discussed earlier, a number of studies have measured levels of the dioxin-like compounds in human breast milk, yielding an average of 35 ppt TEQ_{DFP}-WHO_{98}. Based on a six month nursing scenario, the average daily intake for an infant is about 100 times higher than the adult daily intake on a body weight basis: the adult dose is 1 pg TEQ_{DFP}-WHO_{98}/kg-d, while the infant intake while breast feeding would be about 100 pg TEQ_{DFP}-WHO_{98}/kg-d [(35 pg TEQ_{DFP}-WHO_{98} / g x 800 g/d x 0.04 fat x 0.9 absorption x 365 d/y x 0.5 yr) / (10 kg x 365 d/y x 0.5 yr)]. On a mass basis, the cumulative dose to the infant under this scenario is about 9 percent of the lifetime intake [mass consumed by infant = (100 pg TEQ_{DFP}-WHO_{98}-kd-d x 10 kg x 183 d = 183 ng); mass consumed by adult = (1 pg TEQ_{DFP}-WHO_{98}-kg-d x 70 kg x 69.5 yrs x 365 d/y + 183 ng = 1,960 ng)]. German data on blood serum showed significantly higher levels in nursing infants compared to formula fed infants (Abraham et al., 1995).

Consumption of unusually high amounts of fish, meat, or dairy products containing elevated levels of dioxins and dioxin-like PCBs can lead to elevated blood levels in

comparison to the general population. Most people eat some fish from multiple sources, both fresh and salt water. The typical dioxin concentrations in these fish and the typical rates of consumption are included in the mean background calculation of exposure. People who consume large quantities of fish at typical contamination levels may have elevated exposures since the concentration of dioxin-like compounds in fish are generally higher than in other animal food products. These kinds of exposures are addressed within the estimates of variability of background and are not considered to result in highly exposed populations. If high-end consumers obtain their fish from areas where the concentration of dioxin-like chemicals in the fish is elevated, they may constitute a highly exposed subpopulation. Although this scenario seems reasonable, no supporting data could be found for such a highly exposed subpopulation in the U.S. One study measuring dioxin-like compounds in blood of sports fishers in the Great Lakes area showed elevations over mean background, but within the range of normal variability. Elevated CDD/CDF levels in human blood have been measured in Baltic fishermen. Similarly elevated levels of coplanar PCBs have been measured in the blood of fishermen on the north shore of the Gulf of the St. Lawrence River who consume large amounts of seafood.

Similarly, high exposures to dioxin-like chemicals as a result of consuming meat and dairy products would only occur in situations where individuals consume large quantities of these foods and the level of these compounds is elevated. Most people eat meat and dairy products from multiple sources and, even if large quantities are consumed, they are not likely to have unusually high exposures. Individuals who raise their own livestock for basic subsistence have the potential for higher exposures if local levels of dioxin-like compounds are high. One study in the U.S. showed elevated levels in chicken eggs near a contaminated soil site. European studies at several sites have shown elevated CDD/F levels in milk and other animal products near combustion sources.

Cigarette smokers may also experience increased levels of exposure to CDD/CDFs. This finding is based on data reported by Matsueda et al. (1994) that indicates that the I-TEQ_{DF} content of a pack of cigarettes is approximately 8.6 pg. Assuming that the average smoker smokes 19.1 cigarettes per day, the mean CDD/CDF exposure would be 8.2 pg I-TEQ_{DF}/day. This represents over 10 percent of the average daily background dose of CDD/CDFs for all other sources, as described in Chapter 4.

REFERENCES FOR CHAPTER 5

Abraham, K.; Papke, O.; Ball, M.; Lis, A.; Helge, H. (1995) Changes in blood lipid concentration of PCDDs, PCDFs, and coplanar PCBs in a breast-fed and a formula-fed infant in the second year of life. Organohalogen Compounds. 26:223-225.

Abraham, K.; Palavinskas, R.; Wahn, U.; Helge, H.; Mathar, W. (1998) Still relatively high PCDD/PCDF concentrations in human milk of mothers living in a contaminated area in Germany. Organohalogen Compounds (38):33-36.

Anderson, H.A.; Falk, C.; Hanrahan, L.; Olson, J.; Burse, V.W.; Needham, L.; Paschal, D.; Patterson, D.; Hill, R.H.; Boddy, J.; Budd, M.; Burkett, M..; Fiore, B.; Humphrey, H.E.B.; Johnson, R.; Kanarek, M..; Lee, G.; Monaghan, S.; Reed, D.; Shelley, T.; Sonzogni, W.; Steele, G.; Wright, D. (1998) Profiles of Great Lakes critical pollutants: A sentinel analysis of human blood and urine. Environmental Health Perspectives. 106(5):279-289.

Asplund, L.; Svensson, B.G.; Nilson, A.; Ericksson, U.; Jansson, B.; Jensen, S.; Widegvist, U.; Skerfving, S. (1994) Polychlorinated biphenyls, 1,1,1-trichloro-2,2-bis(p-chlorophenyl) ethane (p,p'-DDT) and 1,1-dichloro-2,2-bis(p-chlorophenyl)-ethylene(p,p'-DDE) in human plasma related to fish consumption. Archives of Environmental Health. 49:477-486.

Ball, D.J.; Creaser, C.S.; Foxall, C.; Lovett, A.A.; MacGuire, F.A.S.; and Patteson, M.T. (1993) Polychlorinated Biphenyls, Dioxins, and Furans in the Pontypool Environment. The Panteg Monitoring Project. Final Report. Centre for Environmental and Risk Management, School of Environmental Sciences, University of East Anglia Norwich NR4 7TJ, UK. Submitted to the Welsh Office of the World Health Organization, April, 1993.

Ball, D.J.; Creaser, C.S.; Foxall, C.; Lovett, A.A.; MacGuire, F.A.S.; and Patteson, M.T. (1994a) Polychlorinated Biphenyls, Dioxins, and Furans in the Pontypool Environment. Research Report No. 21. Centre for Environmental and Risk Management, School of Environmental Sciences, University of East Anglia Norwich NR4 7TJ, UK. Submitted to the Welsh Office of the World Health Organization, January, 1994.

Ball, D.J.; Creaser, C.S.; Foxall, C.; Lovett, A.A.; MacGuire, F.A.S.; and Patteson, M.T. (1994b) Polychlorinated Biphenyls, Dioxins, and Furans in the Pontypool Environment. Research Report No. 22. Centre for Environmental and Risk Management, School of Environmental Sciences, University of East Anglia Norwich NR4 7TJ, UK. Submitted to the Welsh Office of the World Health Organization, October, 1994.

Ball, D.J.; Creaser, C.S.; Foxall, C.; and Lovett, A.A. (1995) Polychlorinated Biphenyls, Dioxins, and Furans in the Pontypool Environment. Research Report No. 25. Centre for Environmental and Risk Management, School of Environmental Sciences, University of East Anglia Norwich NR4 7TJ, UK. Submitted to the Welsh Office of the World Health Organization, June, 1995.

Ball, M.; Päpke, O.; Lis, A. (1990) Polychloridibenzodioxine und polychlordibenzofurane in cigaretterauch. Beiträge zur Tabakforschung International. 14(6):393-402.

Beck, H.; Dross A.; Mathar, W.; and Wittkowsi, R. (1990) Influence of different regional emissions and cardboard containers on levels of PCDD, PCDF, and related compounds in cow milk. Chemosphere. 21: 789-798.

Christmann, W.; Kasiske, D.; Kloppel, K.D.; Partscht, H.; Rotard, W. (1989) Combustion of polyvinylchloride - an important source for the formation of PCDD/PCDF. Chemosphere. 19:387-392.

Cole, D.C.; Kearney, J.; Gilman, A.P.; Ryan, J.J. (1995) Serum PCB, dioxin, and furan levels in Ontario Great Lake anglers. Organohalogen Compounds. 26:193-196.

Columbia River Intertribal Fish Commission (1994) A fish consumption survey of the Umtilla, Nez Perce, Yakima, and Warm Springs Tribes of the Columbia River basin. Technical Report 94-3.

Cooper, K.R.; Fiedler, H.; Bergek, S.; Anderson, R.; Hjelt, M.; Rappe, C. (1995) Polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF) in food samples collected in Southern Mississippi. Organohalogen Compounds. 26:51-57.

Defour, S.; Fraisse, D.; Scherrer, M.C.; Schnepp, B.; Le Querrec, F. (1998) Analysis of polychlorodibenzo-dioxins (PCDDs) and polychlorodibenzo-furans (PCDFs) in milk around industrial sites in France. Organohalogen Compounds. 38:85-88.

De Fre, R.; Wevers, (1998) Measurements of dioxins in deposition and in cow's milk in Belgium. Organohalogen Compounds. 38:113-116.

Dewailly, E.; Ryan, J.J.; Laliberte, C.; Bruneau, S.; Weber, J.P.; Gingras, S.; Carrier, G. (1994) Exposure of remote maritime populations to coplanar PCBs. Environmental Health Perspectives. Vol. 102, Suppl. 1:205-209.

Falk, C.; Hanrahan, L.; Anderson, H.; Kanarek, M.S.; Draheim, L.; Needham, L.; Patterson, D.; and the Great Lakes Consortium (1999) Body burden levels of dioxin, furans, and PCBs among frequent consumers of Great Lakes sport fish. Environmental Research Section A. 80:S19-S25.

Foxall, C.D.; Lovett, A.A.; Creaser, C.S.; Ball, D.J. (1997) Environmental levels of PCDD/DFs and PCBs in the Panteg District of South Wales, UK and their implications for human exposure. Organohalogen Compounds. 31:342-347.

Fries, G.F., Paustenbach, D.J. (1990) Evaluation of potential transmission of 2,3,7,8-tetrachlorodibenzo-p-dioxin-contaminated incinerator emissions to humans via foods. Journal of Toxicology and Environmental Health. 29:1-43.

Hagenmaier, H.; Lindig, C.; She, J. (1994) Correlation of environmental occurrence of polychlorinated dibenzo-p-dioxins and dibenzofurans with possible sources. Chemosphere. 29:2163-2174.

Harrison, N.; de M. Gem, M.G.; Startin, J.R.; Wright, C.; Kelly, M.; Rose, M. (1996) PCDDs and PCDFs in milk from farms in Derbyshire, U.K. Chemosphere. 32:453-460.

Hong, C.S.; Kiao, J.; Casey, A.C.; Bush, B.; Fitzgerald, E.F.; Huang, S.A. (1994) Mono-ortho and non-ortho-substituted polychlorinated biphenyls in human milk from Mohawk and control women: effects of maternal factors and previous lactation. Archives of Environmental Contamination and Toxicology. 27:431-437.

Kjeller, L-O; Jones, K.C.; Johnston, A.E.; Rappe, C. (1991) Increases in the polychlorinated dibenzo-p-dioxin and -furan content of soils and vegetation since the 1840s. Environmental Science and Technology. 25:1619-1627.

Kjeller, L-O; Jones, K.C.; Johnston, A.E.; Rappe, C. (1996) Evidence for a decline in atmospheric emissions of PCDD/Fs in the UK. Environmental Science and Technology. 30:1398-1403.

Liem, A.K.D.; Hoogerbrugge, R,; Kootstra, P.R.; van der Velde, E.G.; de Jong, A.P.J.M. (1991) Occurrence of dioxins in cow's milk in the vicinity of municipal waste incinerators and metal reclamation plant in The Netherlands. Chemosphere. 21:1675-1684.

Löfroth, G.; Zebühr, Y. (1992) Polychlorinated dibenzo-p-dioxins (PCDD) and dibenzofurans (PCDFs) in mainstream and sidestream cigarette smoke. Bulletin of Environmental Contamination and Toxicology. 48:789-794.

Lorber, M.; Pinsky, P.; Gehring, P.; Braverman, C.; Winters, D.; Sovocool, W. (1998) Relationships between dioxins in soil, air, ash, and emissions from a municipal solid waste incinerator emitting large amounts of dioxin. Chemosphere. 37(9-12):2173-2197.

Lovett, A.A.; Foxall, C.D.; Creaser, C.S.; Chewe, D. (1998) PCB and PCDD/F concentrations in egg and poultry meat samples from known urban and rural locations in Wales and England. Chemosphere. 37(9-12):1671-1685.

Luksemburg, W.J.; Mitzel, R.S.; Hedin, J.M.; Silverbush, B.B.; Wong, A.S. (1997) Polychlorinated and dibenzofurans (PCDDs/PCDFs) levels in environmental and human hair samples around a pentachlorophenol plant in China. Organohalogen Compounds. 32:38-40.

Malisch, R. (1998) Increase of PCDD/F contamination in milk and butter in Germany by use of contaminated citrus pulps as component in feed. Organohalogen Compounds. 38:65-70.

Matsueda, T.; Kurokawa, Y.; Nakamura, M.; Takada, S.; Fukamachi, K. (1994) Concentrations of PCDDs, PCDFs and coplanar PCBs in cigarettes from various countries. Organohalogen Compounds. 20:331-334.

Ministry of Agriculture, Fisheries, and Food (MAFF) (1992a) Report of Studies on Dioxins in Derbyshire Carried Out by the Ministry of Agriculture, Fisheries, and Food.

Ministry of Agriculture, Fisheries, and Food (MAFF) (1992b) Third Report of Studies on Dioxins in Derbyshire Carried Out by the Ministry of Agriculture, Fisheries, and Food.

Miyata, H.; Kuriyama, S.; Nakao, T.; Aozasa, O.; Ohta, S. (1998) Contamination levels of PCDDs, PCDFs and non-ortho coplanar PCBs in blood samples collected from residents in high cancer-causing area close to batch-type municipal solid waste incinerator in Japan. Organohalogen Compounds. 38:143-146.

Moche, W.; Thanner, G. (1997) Ambient air patterns and concentrations of PCDD/F in the vicinity of steelworks in Austria. Organohalogen Compounds. 32:380-383.

National Academy of Sciences (NAS) (1991) Nutrition during lactation. Washington, DC: National Academy Press.

National Center for Health Statistics (1987) Anthropometric reference data and prevalence of overweight, United States, 1976-1980. Data from the National Health and Nutrition Examination Survey, Series 11, No. 238. Hyattsville, MD: U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics. DHHS Publication No. (PHS) 87-1688.

Ohio Environmental Protection Agency (OEPA) (1994) Risk assessment of potential health effects of dioxins and dibenzofurans emitted from the Columbus solid waste authority's reduction facility. The Ohio Environmental Protection Agency, Division of Air Pollution Control. February 28, 1994.

Ohta, S.; Kuriyama, S.; Nakao, T.; Aozasa, O.; Miyata, H.; Tanahaski, M. (1997) Levels of PCDDs, PCDFs, and non-ortho coplanar PCBs in soil collected from high cancer-causing area close to batch-type municipal solid waste incinerator in Japan. Organohalogen Compounds. 32:155-160.

Rappe, C.; Nygren, M.; Lindstrom, G.; Buser, H.R.; Blaser, O.; Wuthrich, C. (1987) Polychlorinated dibenzodioxins and dibenzo-p-dioxins and other chlorinated contaminants in cow milk from various locations in Switzerland. Environmental Science and Technology. 21:964-970.

Reed, L.W.; Hunt, G.T.; Maisel, B.E.; Hoyt, M.; Keefe, D.; and Hackney, P. (1990) Baseline assessment of PCDDs/PCDFs in the vicinity of the Elk River, Minnesota generating station. Chemosphere. 21:159-171.

Riss, A. (1993) Impact of PCDD/PCDF emissions from a copper reclamation plant: five years of experience with environmental monitoring. Proceedings of Dioxin 93, held in Vienna, Austria, September, 1993. Organohalogen Compounds. 14:23-26.

Riss, A.; Hagenmaier, H.; Weberruss, U.; Schlatter, C.; Wacker, R. (1990) Comparison of PCDD/PCDF levels in soil, grass, cow's milk, human blood and spruce needles in an area of PCDD/PCDF contamination through emissions from a metal reclamation plant. Chemosphere. 21:1451-1456.

Sandalls, J.; Berryman, B.; Bennett, L.; Newstead, S.; Fox, A. (1998) PCDD/PCDF on land around an industrial site and identification of source. Chemosphere. 37(9-12):2225-2237.

Schecter, A.; Papke, O. (1998) Dioxins, dibenzofurans, and coplanar and marker PCBs in blood of persons residing near a PCB manufacturing facility in the United States. Organohalogen Compounds. 38:175-178.

Smith, A.H. (1987) Infant exposure assessment for breast milk dioxins and furans derived from waste incineration emissions. Risk Analysis. 7(3):347-353.

Startin, J.R.; Wright, C.; Kelly, M.; Charlesworth, E.A. (1994) Dioxin concentrations in the blood of individuals resident on farms near Bolsover U.K. Organohalogen Compounds. 21:117-120.

Sullivan, M.J.; Custance, S.R.; Miller, C.J. (1991) Infant exposure to dioxin in mother's milk resulting from maternal ingestion of contaminated fish. Chemosphere. 23(8-10):1387-1396.

Svensson, B.G.; Nelsson, A.; Hansson, M.; Rappe, C.; Akesson, B.; Skerfving, S. (1991) Exposure to dioxins and dibenzofurans through the consumption of fish. New England Journal of Medicine. 324(1):8-12.

U.S. Department of Agriculture (1995) Food and nutrient intakes by individuals in the United States, 1 day, 1989-91. Washington, DC: Agricultural Research Service. NFS Report No. 91-2.

U.S. Environmental Protection Agency (1992) Respiratory health effects of passive smoking: lung cancer and other disorders. Washington, DC: Office of Research and Development. EPA/600/6-90/006F.

U.S. Environmental Protection Agency (1996) Health Assessment for 2,3,7,8-TCDD and related compounds, Washington, DC: Office of Research and Development. EPA/600/BP-92/001.

U.S. Environmental Protection Agency (1997) Exposure Factors Handbook. Washington, DC: Office of Research and Development. EPA/600/P-95/002B.

U.S. Environmental Protection Agency (1999) Risk assessment guidance for Superfund Volume I: Human health evaluation manual (Part E, supplemental guidance for dermal risk assessment) interim guidance, draft. Washington, D.C. Office of Emergency and Remedial Response.

Winters, D.; Cleverly, D.; Meier, K.; Dupuy, A.; Byrne, C.; Deyrup, C.; Ellis, R.; Ferrario, J.; Harless, R.; Leese, W.; Lorber, M.; McDaniel, D.; Schaum, J.; Walcott, J. (1996) A statistical survey of dioxin-like compounds in United States beef: A progress report. Chemosphere. 32:469-478.

Wolfe, R.J.; Walker, R.J. (1987) Subsistence economics in Alaska: productivity, geography and developmental impacts. Arctic Anthropology. 24(2):56-81.

Table 5-1. Concentrations of CDDs, CDFs, and Dioxin-Like PCBs in Blood (lipid based) of a Breast-Fed and a Formula-Fed Infant at the Age of 11 and 25 Months

Compound (conc. in pg/g fat)	Age (Months)			
	Breast-Fed Infant		Formula-Fed Infant	
	11	25	11	25
2,3,7,8-T4CDF	< 2.7	< 2.5	< 3.0	< 2.5
2,3,7,8-T4CDD	3.7	4.1	< 1.0	< 1.0
1,2,3,7,8-P5CDF	< 1.2	n.d. (1.4)	< 1.2	n.d. (2.5)
2,3,4,7,8-P5CDF	23.1	29.7	1.5	< 2.5
1,2,3,7,8-P5CDD	11.1	15.2	< 1.0	n.d. (1.8)
1,2,3,4,7,8H6CDF	9.8	12.2	< 2.2	< 2.5
1,2,3,6,7,8-H6CDF	8.1	10.2	< 1.0	< 2.5
2,3,4,6,7,8-H6CDF	< 3.4	< 3.0	< 2.3	< 2.5
1,2,3,4,7,8-H6CDD	7.8	9.1	n.d (1.1)	n.d. (2.8)
1,2,3,6,7,8-H6CDD	43.0	51.7	2.5	< 5.4
1,2,3,7,8,9-H6CDD	7.1	8.1	n.d. (1.2)	< 4.5
1,2,3,4,6,7,8-H7CDF	13.1	n.a.	< 5.8	< 6.0
1,2,3,4,6,7,8-H7CDD	24.3	29.7	8.8	< 10.0
OCDF	< 5.0	n.a.	< 5.0	n.a.
OCDD	148.7	204.0	79.3	70.0
I-TEQ (< LD = 0.5*LD)	29.2	36.8	2.4	2.3
PCB 77	23 (m)	20 (m)	26 (m)	20 (m)
PCB 126	287	n.a.	24	n.a.
PCB 169	270	183	7	11

n.a. = not analyzed
n.d. = not detected (limit of detection)
(m) = maximum value, due to possible contribution of a contaminant

Source: Abraham et al. (1995).

Table 5-2. Estimated CDD/CDF Exposures for Adult Subsistence Fishermen

Media	Conc. TEQ_{DF}-WHO_{98}[a]	Contact Rate[b]	Daily Intake (pg/kg-day)[c]
Soil ingestion	11.9 ppt[e]	50 mg/day	8.5×10^{-3}
Soil dermal contact	11.9 ppt	12 g/day[f]	2.0×10^{-3}
Freshwater fish ingestion	1.2 ppt[g]	59 to 170 g/day	$2.9 \times 10^{+0}$
Marine fish ingestion	0.36 ppt	12.5 g/day	6.4×10^{-2}
Marine shellfish ingestion	0.79 ppt	1.6 g/day	1.8×10^{-2}
Inhalation	0.12 pg/m^3	13.3 m^3/day	2.3×10^{-2}
Water ingestion	0.00056 ppq	1.4 L/day	1.1×10^{-5}
Milk ingestion	0.031 ppt	175 g/day	7.8×10^{-2}
Dairy ingestion	0.12 ppt	55 g/day	9.4×10^{-2}
Vegetable fat ingestion	0.056 ppt[e]	17 g/day	1.4×10^{-2}
	Total		1.3 to $3.2 \times 10^{+0}$ [d]

[a] Values from Table 3-54.

[b] Values for adult soil ingestion, inhalation, water ingestion, and subsistence fish ingestion from Exposure Factors Handbook (U.S. EPA, 1997). Contact rates for milk, dairy, and vegetable fats are based on data from USDA (1995).

[c] Daily intake (mg/kg-day) = [Contact rate (g/day; m^3/day; L/day; mg/day) x Conc. TEQ x Unit Conversion (soil unit conversion = 10^{-3}, all other media no unit conversion needed)/Body Weight (kg)] or Contact rate (g/kg-day) x Conc. TEQ x Unit Conversion.

[d] Approximately equivalent to 92 to 225 pg/day, assuming an adult body weight of 70 kg.

[e] Calculated by setting nondetects to zero.

[f] Calculated as the surface area of the body that contacts the soil (5,700 cm^2/day) x the rate that soil adheres to the skin (0.07 mg/cm^2) x the fraction of CDD/CDFs absorbed through the skin (0.03); exposure factors based on recommendations in U.S. EPA (1999) for an adult resident, which assumes that the lower legs, forearms, hands, and head are exposed to the soil.

[g] Represents I-TEQ_{DF}. TEQ_{DF}-WHO_{98} could not be calculated because congener-specific data were not available.

Table 5-3. Levels of Different PCB Congeners in Blood Samples from Three Groups of Men with Different Fish Consumption Habits

Congener (UIPAC)	Fish Intake					
	None		Moderate		High	
	Plasma (n = 9)	Lipid (n = 8)	Plasma (n = 14)	Lipid (n = 7	Plasma (n = 14)	Lipid (n = 11)
Non-ortho-PCBs						
77 (pg/g)[a]	0.04 (0.01-0.09)	15 3-38)	0.1[b] (9.03-0.2)	41[b] (26-62)	0.2[b,c] (0.1-0.5)	50[b] (15-140)
126 (pg/g)	0.73 (0.3-1.2)	220 (100-450)	1.05 (0.6-2.4)	400[b] (210-650)	2.8[b,c] (1.2-4.9)	790[b,c] (380-1400)
169 (pg/g)	0.65 (1.3-1.5)	200 (100-340)	0.86 (0.4-1.7)	250 (170-360)	1.80[b,c] (0.3-3.6)	570[b,c] (210-1200)
Mono-ortho-PCBs						
105 (ng/g)	0.02 (0-0.03)	5 (0-13)	0.04 (0.02-0.07)	14[b] (9-20)	0.14[b,c] (0.04-0.3)	39[b,c] (18-77)
118 (ng/g)	0.12 (0.05-0.21)	41 (17-92)	0.21 (0.12-0.43)	76 (45-120)	0.58[b,c] (0.21-1.00)	160[b,c] (84-300)
156 (ng/g)[d]	0.13 (0.05-0.34)	40 (19-68)	0.14 (0.07-0.28)	44 (30-64)	0.3[b,c] (0.05-0.7)	90[b,c] (36-180)
157 (ng/g)[d]	0.02 (0.01-0.05	6.6 (2.8-11)	0.02 (0.01-0.05)	7.8 (5.4-11)	0.06[b,c] (0.01-0.14)	18[b,c] (7.4-39)
Di-ortho- and other PCBs						
180 (ng/g)[e]	1	400	1	400	2	600

Notes: Means and ranges indicated on plasma and lipid basis.

[a] Near the detection limit.
[b] $p < .05$, compared with group "none."
[c] $p < .05$, compared with group "moderate."
[d] Quantified, using single-response factors.
[e] CB-180 quantified from two fractions, concentrations thus estimated.

Source: Asplund et al. (1994).

Table 5-4. Mean TEQ Levels in Pooled Serum Samples

	$I\text{-}TEQ_{DF}$ (ppt, lipid basis)	$TEQ_P\text{-}WHO_{94}$ (ppt, lipid basis)
Cornwall		
Sports Fishers		
< 38 years, lower	20.8	--
higher	22.2	3.6
38 years, lower	28.4	3.1
higher	31.4	9.5
> 50 years, higher	33.5	17.3
Nonfish Eaters		
< 38 years	24.7	2.6
38-50 years	29.8	6.8
> 50 years	36.8	9.7
Mississauga		
Sports Fishers		
< 38 years	32.4	--
38-50 years	40.1	--
> 50 years	41.2	--
Nonfish Eaters		
< 38 years	34.0	--
38-50 years	29.1	--
> 50 years	34.3	--

Source: Adapted from Cole et al. (1995).

Table 5-5. Mean CDD/CDF Levels in Serum of Consumers of Great Lakes Sport Fish (ppt, lipid adjusted)

	All Sport Fish Consumer Subjects (ppt) (n = 31)[a]	Lake Michigan Participants (ppt) (n = 9)	Lake Huron Participants (ppt) (n-11)	Lake Erie Participants (ppt) (n = 11)	Comparison Group[a] (ppt) (n = 70)
CDD Congeners					
2,3,7,8-TCDD	5.6	4.7	10.5	4.9	2.8
1,2,3,7,8-PeCDD	10.4	9.8	16	5.8	5.5
1,2,3,4,7,8-HxCDD	8.4	11.4	8.4	5.5	9.0
1,2,3,6,7,8-HxCDD	126	120	142	115	70.8
1,2,3,7,8,9-HxCDD	7.0	8.7	5.5	5.8	8.4
1,2,3,4,6,7,8-HpCDD	134	144	153	95.9	124
1,2,3,4,6,7,9-HpCDD		ND	ND		4.4
OCDD	777	783	918	623	971
Total	1,062	1,087	1,258	844	1,188
Total I-TEQ_D	27.5	25.8	36	20.7	15.5
CDF Congeners					
2,3,7,8-TeCDF	2.2	2.4	2.1		2.1
1,2,3,7,8-PeCDF	2.0	ND	1.7	ND	1.6
2,3,4,7,8-PeCDF	17.7	20.4	22.8	10.4	5.5
1,2,3,4,7,8-HxCDF	12.7	11.6	16.0	10.2	8.0
1,2,3,6,7,8-HxCDF	9.0	8.0	10.5	7.7	5.3
1,2,3,7,8,9-HxCDF	ND	ND	ND	ND	1.8
2,3,4,6,7,8-HxCDF	5.1	6.0	4.8	8.0	3.8
1,2,3,4,6,7,8-HpCDF	20.0	22.1	22.9	15.2	21.3
1,2,3,4,7,8,9-HpCDF	ND	ND	ND	ND	NA
OCDF		ND		ND	6.9
Total	58.2	70.8	79.3	48.3	87.3
Total I-TEQ_F	11.9	13.2	14.8	7.8	4.9

a One individual was excluded from the data summary due to unusually high occupational/environmental exposures.
b Comparison group is from a 1991 unpublished NCEH/CDC data set of a Jacksonville, Arkansas, population of 70 individuals.

Source: Anderson et al. (1998).

Table 5-6. Mean PCB Levels in Serum of Consumers of Great Lakes Sport Fish (ppt, lipid adjusted)

	All Sport Fish Consumer Subjects[a] (ppt) (n = 31)	Lake Michigan Participants (ppt) (n = 9)	Lake Huron Participants (ppt) (n-11)	Lake Erie Participants (ppt) (n = 11)	Comparison Group[b] (ppt) (n = 70)
Coplanar PCB Congeners					
77	14.6	16.5	14.2	13.3	12.6
81	13.5	17.4	13.2		8.6
126	148	261	187	28	18.4
169	80.8	113	84.2	48.4	17.9
Coplanar PCB Total	228	340	282	75.4	57.4
$I\text{-}TEQ_{P}\text{-}WHO_{94}$	17.4	26	23	4.8	1.8
Congener-Specific PCBs					
28	0.08	0.08	0.1	0.08	ND
52	0.01	ND	0.01	ND	ND
56	0.02	0.06	ND	ND	ND
58	0.04	0.08	0.04	0.01	ND
74	0.3	0.6	0.4	0.2	0.009
99	0.4	0.7	0.5	0.1	ND
101	ND	0.01	ND	ND	ND
1.5	0.1	0.2	0.1	0.02	0.4
118	0.4	0.8	0.5	0.08	0.03
130	0.1	0.2	0.1	0.02	NA
138	0.8	1.3	0.8	0.4	0.4
146	0.2	0.3	0.2	0.04	ND
153	1.1	1.7	1.1	0.6	0.4
156	0.02	0.04	0.02	ND	NA
157	0.1	0.2	0.1	0.08	NA
187	0.03	0.07	0.03	ND	ND
170	0.1	0.2	0.2	0.07	ND
172	0.02	0.05	0.03	ND	ND
177	0.04	0.09	0.06	ND	ND
178	0.07	0.13	0.08	0.03	ND
180	0.4	0.8	0.4	0.2	0.4
183	0.1	0.2	0.1	0.03	ND
187	0.3	0.4	0.3	0.08	0.04
189	ND	ND	ND	ND	NA
193	0.03	0.08	0.02	ND	NA
194	0.09	0.1	0.1	0.05	0.004
195	0.04	0.07	0.06	ND	ND
201	0.2	0.3	0.2	0.09	0.04
203/196	0.09	0.2	0.11	0.03	0.007
206	0.07	0.08	0.08	0.04	ND
209	0.02	0.03	0.04	ND	NA
Total	5.2	8.6	5.7	2.2	1.2

a One individual was excluded from the data summary due to unusually high occupational/environmental exposures.
b Comparison group from a 1996 unpublished data set of 41 non-Great Lake sport fish consumers analyzed by the Wisconsin State Laboratory of Hygiene.

Source: Anderson et al. (1998).

Table 5-7. Comparison Between Mean PCB Levels in Fish-eating Populations and Controls

PCBs	Fishermen		Controls	
	Mean Concentration (ppt, lipid basis)	TEQ_P-WHO_{94} (ppt, lipid basis)	Mean Concentration (ppt, lipid basis)	TEQ_P-WHO_{94} (ppt, lipid basis)
126	1540	154	48	4.8
169	1010	10.1	29	0.29
118	568	56.8	25.4	2.54
170	539	53.9	27.7	2.77
180	1776	17.76	48.2	0.48
TOTAL TEQ_P-WHO_{94}	--	292.6	--	10.9

Source: Adapted from Dewailly et al. (1994)